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Short report

Electroencephalographic abnormalities in antisocial personality disorder

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A B S T R A C T

The presence of brain dysfunction in violent offenders has been frequently examined with inconsistent results. The aim of the study was to assess the EEG of 84 violent offenders by visual inspection and frequency-domain quantitative analysis in 84 violent prisoners. Low-resolution electromagnetic tomography (LORETA) was also employed for theta band of the EEG spectra. Antisocial personality disorder (ASPD) was present in 50 of the offenders and it was absent in the remaining 34. The prevalence of EEG abnormalities, by visual inspection, was similar for both the ASPD group (82%) and non-ASPD group (79%). The brain topography of these anomalies also did not differ between groups, in contrast to results of the EEG quantitative analysis (QEEG) and LORETA that showed remarkable regional differences between both groups. QEEG analysis showed a pattern of excess of theta-delta activities and decrease of alpha band on the right fronto-temporal and left temporo-parietal regions in the ASPD group. LORETA signified an increase of theta activity (5.08 Hz) in ASPD group relative to non-ASPD group within left temporal and parietal regions. Findings indicate that QEEG analysis and techniques of source localization may reveal differences in brain electrical activity among offenders with ASPD, which was not obvious to visual inspection.

1. Introduction

Antisocial personality disorder (ASPD) is a psychiatric diagnosis[1,2] related to criminal and violent behaviour.[3,4] Although the contribution of neurobiological factors to this personality disorder remains uncertain,[5] anatomical and functional cerebral impairments, that could be directly or indirectly involved in the expression of violent behaviour have been described in several studies.[6–10] In a previous study we described electroencephalographic abnormalities in a group of 18 subjects suffer from ASPD.[10] Here, we are presenting the results from a larger group of violent behaviour offenders. The study aimed to determine the presence of electrophysiological differences between violent offenders with and without ASPD, specifically we wanted to re-assess whether the results reported in our previous study[10] could be explain by the implicit heterogeneity of ASPD diagnosis.

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2. Materials and methods

2.1. Subjects

The study included 84 violent male offenders, mean age was 30.3 years (SD = 8.47) from a prison located in Havana City, serving sentences for committing violent criminal acts (homicides or murders). Assessment was conducted during a 2 years period, from January 2004 to December 2005.

The psychiatric diagnosis was made using offenders clinical findings and institutional files, which included personal and educational history, past history of drugs use, mental status, the results of a Diagnostic and Statistical Manual of mental disorders, DSM-IVR (4th ed. Revised) and the structured clinical interview performed by a trained psychiatrist.[11] All offenders scored within the normal intelligence range on the Wechsler Adult Intelligence Scale-Revised (WAIS-R).[12] Fifty violent offenders were diagnosed with ASPD (mean age = 30.2, SD = 8.13 years). Offenders diagnosed with ASPD, fulfilled the DSM-IVR criteria, of the onset of the violent behaviour patterns before the age of 15-year-old and comprised at

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least three of the following behaviours: repeated criminal acts, deceitfulness, impulsiveness, recurrent fights or assaults, disregard for other persons' safety, irresponsibility and lack of remorse. The non-ASPD group was composed of 34 violent offenders that did not fulfil DSM-IVR criteria for any personality disorder diagnosis (mean age = 30.4, SD = 9.15 years).

The study inclusion criteria considered, with the exception of ASPD, the lack of any current or previous history of neurological or psychiatric diseases. Alcoholics or any other drug addiction were not included. All the offenders were medication free at the time of testing. Written informed consent was obtained from all offenders (the data from five additional offenders were excluded from the final analysis, because they opted to withdraw from the study after their participation had begun). The study was approved by the Ethics Committee of the Cuban Center of Neurosciences.

2.2. EEG recording

EEGs were recorded using a 21-channel MEDICID IV EEG system (Neuronic S.A., Havana). Surface electrodes were placed at 19 sites of the International 10–20 system[13] (Fp1, Fp2, Fz, F3, F4, F7, F8, Cz, C3, C4, T3, T4, T5, T6, Pz, P3, P4, O1 and O2), and referenced to linked ear lobes. Electrodes impedance was equal or less than 5 kΩ. EEG was amplified by 10,600, with a bandpass from 0.5 to 30 Hz and sampled through a 12-bit analogue-to-digital converter at 200 Hz. The EEG was recorded in a temperature and noise controlled room while the participant was lying on a bed. All individuals were asked to relax and remain at rest during the test in order to minimize artifacts produced by movements, and also to avoid excessive blinking.

Each resting EEG was obtained during eight to 10 min with closed eyes. Subsequently, 2 min of alternation between close and open eyes, following 3 min carrying out hyperventilation, and then 2 min of recovery were also recorded. Taking into account that sleepiness could have caused an enhancement of theta activity, the individual vigilance level was checked during EEG acquisition, seeking for slowing of the EEG background activity or for the appearance of typical sleep patterns. In addition, at the end of the recording process, individuals were asked about whether they were awake during the whole recording session.

2.3. Visual assessment of the EEG

Several bipolar montages were used for off line EEG interpretation. The EEG was considered normal if it had adequate organization of the background activity (according to the individual age), a well-defined spatial differentiation, rhythmic alpha activity and absence of slow or paroxysmal activity.[14] Slow EEG activity was defined as the presence of persistent nonrhythmic theta-delta slow waves. Paroxysmal EEG activity included spikes, sharp waves, and spike and slow wave complexes. EEGs presenting both types of previously described abnormalities were included in the slow and paroxysmal category. Ratios and percentages in all categories were calculated.

2.4. Quantitative EEG analysis (QEEG)

Selection of EEG segments for QEEG analysis was done by visual inspection, and segments containing artifact (i.e. eye movements, eye blinks, muscle activity, or other artifacts) were excluded. For this reason, it was only possible to obtain 20–24 closed eyes state segments (without artifact) of 2.56 s from each individual for quantitative EEG analysis. The exact number of segments depended on how cooperative the individual was, getting a minimum of 20 required for the study entry. One minute of artifact-free EEG is considered the minimum amount of EEG required to obtain reliable quantitative measures.[15,16] Fast Fourier Transform (FFT) was applied in order to obtain the cross spectral matrixes of all individual records[17] which were calculated with a spectral resolution of 0.39 Hz, from 0.78 to 19.53 Hz.

Quantitative measures were log-transformed to obtain a normal distribution. All spectral measures were compared to gender and age-matched normative database using Z-scores (see the statistics section for database details).

2.5. EEG source estimation

The Low-Resolution Electromagnetic Tomography (LORETA)[18] was used to compute, from the scalp-recorded electric potential distribution, the three-dimensional distribution of electrical activity (i.e., the current density) produced by neuronal generators within a three-shell spherical head model. The head model includes scalp, skull, and brain compartment. The brain compartment was coregistered to the Talairach probability brain atlas, digitized at the Brain Imaging Center of the Montreal Neurologic Institute and consisted of 2394 voxels at 7 mm spatial resolution. The LORETA functional images represent the electrical activity at each voxel as squared magnitude (i.e., power) of computed current density.

Using the Neuronic QEEG analysis software (Neuronic S.A.), selected EEG frequency range (theta band) was saved in a "text" format to be read later on through a specific software system developed for this purpose. Current density vectors (CD) were calculated for each individual from all the data segments using the Neuronic Source Localizer software (Neuronic S.A.). This program provided a spatially restricted solution to cortical gray matter and basal ganglia in the Talairach Human Brain Atlas.

2.6. Statistical analysis between groups

In order to determine the dependence among categorical variables, Chi square test, exact F Fischer and McNemar chi-squared were used for analysis (Statistic 6.0 for Windows). The level of statistical significance was set at 0.05 for all the tests.

In order to compare the present data with our previously published results, two proportion tests were used. The equation for the test was:

$$|t| = \sqrt{[(N_1 * N_2)/(N_1 + N_2)]} * |p_1 - p_2| / \sqrt{(p * q)}$$

where N_i is the sample size of the group, pi is the proportion of the group.

$$p = (p_1 * N_1 + p_2 * N_2)/(N_1 + N_2)$$

$$q = 1 - p$$

The degrees of freedom were computed as

$$N_1 + N_2 - 2$$

2.6.1. QEEG analysis Z spectrum

The mean of EEG cross-spectral parameters for both ASPD and non-ASPD groups were compared with the Cuban normative database using the Z transform.[19] This normative database was constructed from the EEG of 211 normal subjects' (105 males, 106 females) with an age range from 5 to 97 years. Normative coefficients were obtained by carrying out a polynomial regression with

age of each log spectral value. Normalized values, expressed as the number of standard deviations from the mean of the norm, were calculated for every frequency and electrode and stored as a "*Z* spectrum".[19] Factors like age might affect EEG data by increasing inter-individual variability.[20] The use of normalized values for statistical analysis eliminates these effects that, otherwise, should have been taken into account for comparisons between the groups.

2.7. Statistical methodology to compare the Z spectra mean of both groups

In order to evaluate differences between the *Z* spectra of both groups, a permutation test was used.[21–24] The permutation test has the following advantages: free distribution − which controls the experiment wise error for simultaneous univariate comparisons. No assumption of an underlying correlation structure. Providing exact *p*-values for any number of individuals, frequency points and recording sites.

The t statistics and max (t) were calculated. Max (t) represented the maximum of t statistic in each electrode, and frequency.

Multivariate statistics can be used to summarize and test differences between two Z spectra obtained from the maximum value of all the univariate statistics.

These statistics were obtained as follows

Step 1: the observations were repeatedly permutated between groups. Both statistics were calculated for every repetition.

Step 2: the distribution was estimated using the statistics calculated in the step above.

Step 3: significance levels was set using the t and max(t) of the original samples with the distribution estimated in Step 2.[21–24]

2.7.1. Inverse solution analysis

In order to identify significant regional differences between groups in current density (CD) for slow EEG bands, a t-test for independent samples with correction for multiple comparisons was performed (Neuronic Statistica software, Neuronic S.A.). The final outcome was a map of the t-test values for each voxel thresholded at a false discovery rate (FDR) $q = 0.2$. Coordinates of main activation are represented in Talairach space (Neuronic Tomographic Viewer, Neuronic, SA).

3. Results

3.1. Visual inspection

Table 1 presents details of the obtained outcomes of the EEG visual evaluation. They were very similar for both groups. The prevalence of EEG anomalies subtypes did not differ between the two groups (Pearson chi-square = 0.23, df = 3, $p < 0.97$).

Table 2 shows the topographical distribution of the EEG abnormalities found in ASPD and non-ASPD groups. Over 30% of the cases presented widespread EEG abnormalities. The frontal and temporal lobes were the most affected brain regions due to slow EEG alterations, whereas paroxysmal activity mostly affected the frontal region. There were no groups differences in topographical distribution of slow EEG activity (Pearson chi-square = 0.41, df = 2, $p < 0.81$).

When groups were confronted, statistically significant differences were not found (total frontal abnormalities × groups − Fisher: $p < 0.50$; total temporal abnormalities × groups − Fisher $p < 0.31$).

In spite of the homogeneous topographical distribution of these EEG abnormalities in both groups, the proportions of frontal and temporal slow and paroxysmal activities were higher in ASPD group (frontal slow activity McNemar chi-square = 7.31; $p < 0.01$; temporal slow activity McNemar chi-square = 27.7, $p < 0.001$; frontal paroxysmal activity McNemar chi-square = 22.1, $p < 0.001$; temporal paroxysmal activity McNemar chi-square = 29.6, $p < 0.001$).

Comparisons between our previous reported data[10] and the current outcomes, with regard to different visual assigned EEG categories were carried out using a proportion test. Significant difference regarding the slow EEG presence ($p < 0.05$) was found. Present ASPD group showed less slow EEG activity and statistically significant increase of paroxysmal activity ($p < 0.001$) when it was compared to previous research.

Table 1
Classification of the subject's EEG in both groups by visual inspection.

Group	Normal	Slow	Paroxysmal	Slow and paroxysmal
ASPD	9 (18%)	24 (48%)	8 (16%)	9 (18%)
Control	7 (20.6%)	15 (44.1%)	5 (14.7%)	7 (20.6%)

3.2. Quantitative EEG analysis

Using the permutation test, statistically significant differences were found for the mean of the parameters of cross-spectral measurements between ASPD and non-ASPD groups in the following regions and frequencies:

- Delta band (1.17 Hz) on the left parietal region,
- Theta band within a frequency range of 3.52–7.41 Hz, on bilateral temporal regions and the left central lead,
- Beta band within a frequency range of 15.23–18.75 Hz on the right frontal-temporal areas and the left temporal region.

The power values for these frequencies were increased for the ASPD group, but on the contrary, for frequency range of the alpha band within the 7.81–13.28 Hz, a decrease of the energy was found on the right frontal-temporal and left temporal-parietal regions for the ASPD group.

3.3. EEG generator analysis

LORETA source imaging revealed a significant increase of theta activity at 5.08 Hz on the following Brodmann's areas: 21 (left middle temporal gyrus); 20 (left inferior temporal gyrus); 37 (left fusiform gyrus); 48 (left retrosubicular); 41 and 42 (left Heschl gyrus); 43 (left postcentral area); 38 (left superior temporal pole); 39 (left gyrus angular); 2 and 3 (left postcentral gyrus) and 40 (left inferior parietal lobule) in the ASPD group when they were compared with offenders without the psychiatric disorders ($p < 0.05$ after false discovery rate correction for multiple comparisons; Fig. 1)

4. Discussion

In previous study, the examination of the electrophysiological characteristics in smaller ASPD group found slow wave activity with abnormalities of the background activity in most of the individuals. Contrary to our predictions, concerning the type of abnormalities present in the visual analysis of the EEG or its localization, in this research, there was no found any significant difference between ASPD and non-ASPD violent offenders; however significant differences in regional current source density

Table 2
EEG abnormalities topographic distribution

Group	Slow				Paroxysmal		Slow and paroxysmal		
	Frontal	Temporal	Parietal	Widespread	Frontal	Temporal	Frontal	Temporal	Widespread
ASPD	4 (8%)	4 (8%)	1 (2%)	15 (30%)	7 (14%)	1 (2%)	4 (8%)	1 (2%)	4 (8%)
Control	2 (5.9%)	1 (2.9%)	3 (8.8%)	9 (26.5%)	4 (11.8%)	1 (2.9%)	3 (8.8%)	0 (0%)	4 (11.8%)

were found between these groups when LORETA analysis was performed.

Slow wave abnormality was present in offender groups, ASPD and the non-ASPD. It suggests that this type of abnormality may be related to the central nervous system (CNS) dysfunction present in many types of violent behaviours.[25]

Although both, generalized and focal patterns of abnormal function were observed, frontal and temporal cortical regions showed higher numbers of slow and paroxysmal abnormalities. The number of offenders with these EEG alterations was higher in the ASPD group. These findings demonstrate that dysfunction in these regions play an important role in ASPD diagnosis. Frontal lobes are crucial for the so-called executive functions of anticipation, goal selection, planning, self-monitoring, use of feedback, and completion of purposeful activities. They are important indeed for selection and control of socially relevant behaviour.[25–28] When this function is impaired, all the other cognitive systems are affected. Since frontal lobes regulate the abilities to control impulses, reason and make socially responsible judgments, injury to these lobes may cause violent and aggressive behaviour.

Temporal lobes take part in sensory, affective, and higher cognitive processing.[29,30] If those areas do not function properly, a person may act impulsively and inappropriately. The associated inability to act in a “civilized” manner, so often results in augment of criminality.[25–28]

Independently of the sleepiness deprivation is one of the variables may influence on the origin of EEG-slowing,[31,32] we considered that the excess of slow activity found in this study did not depend on this factor. In the current research all offenders, prior to EEG recording, had a psychiatry assessment and were questioned

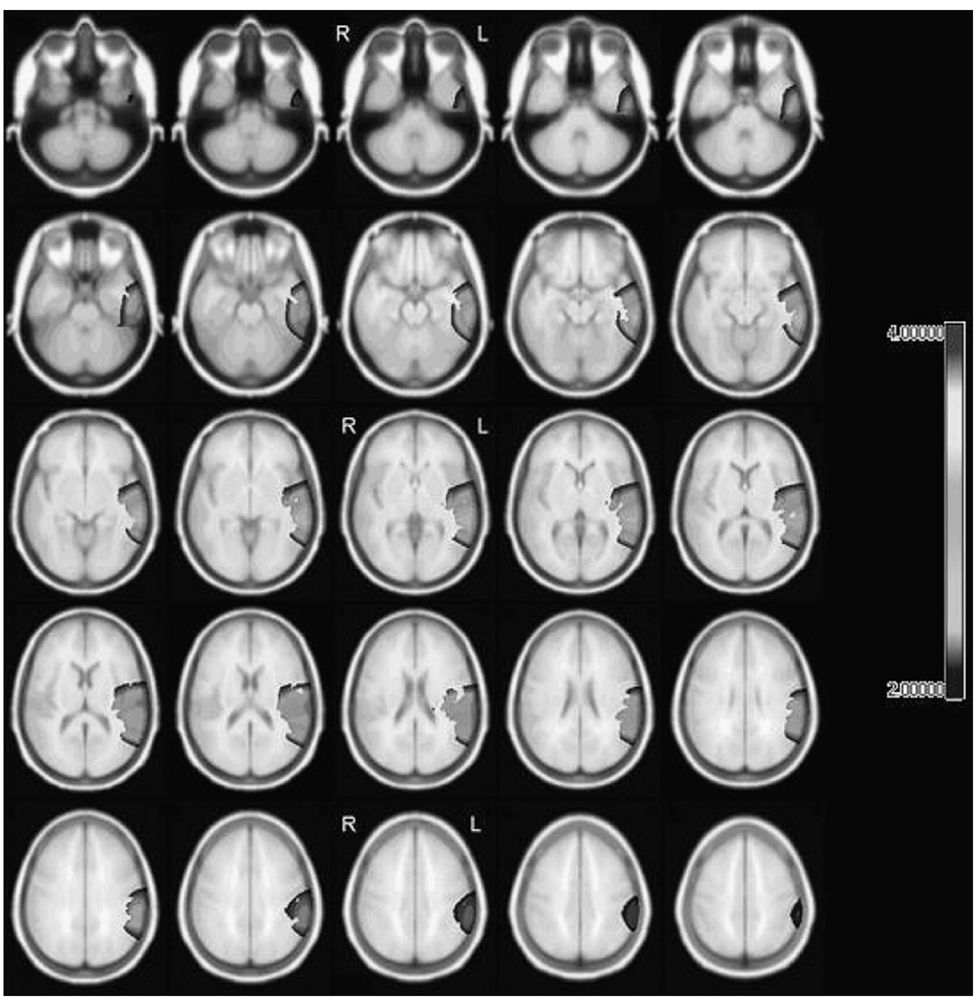

Fig. 1. Anatomical distribution of maximum t values between ASPD and Control groups (q-value: 0.2, after false discovery rate correction for multiple comparisons). The highest significant differences were found on the left hemisphere, mainly increase of theta activity on the left middle temporal gyrus, inferior temporal gyrus, left fusiform gyrus, left retrosubicular, left heschl gyrus, left postcentral area, left superior temporal pole, left gyrus angular, postcentral gyrus and left inferior parietal lobule. R, right hemisphere; L, left hemisphere.

about the quantity and quality of sleep. None of them referred clinical signs or symptoms associated to sleeping disorders; moreover during the EEG recording was controlled the presence of any electrophysiological signs related to the tendency to fall asleep in order to rule out this influence in our electroencephalographic findings.

Data from other studies have suggested that nicotine and caffeine consumption could be associated with changes in EEG activity.[33–35] Increase of alpha power had been reported as a nicotine effect in rest EEG with closed eyes.[34,35] A decrease in alpha activity was found in this study. The result is consistent with findings from other studies where violent individuals had been involved.[36] Therefore it is probable that alpha decreasing observed may be higher without the influence of the nicotine over the EEG activity. With regard to caffeine, studies designed to characterize the effects of caffeine on EEG had showed increase of beta power,[35,37,38] as the outcome of this research found in the ASPD group. Nevertheless, there is a very low level of consumption of this legal drug during the permanence in prison (at least in Cuba), that is why it is considered that caffeine effects were not responsible for the increase of beta power in the ASPD group.

Electroencephalographic measurements have produced laterality effects in different studies, however the majority of them have not evaluated the resting EEG or analyzed EEG frequency bands separately.[39–41] Unfortunately in this study handedness was not controlled. Some studies in violent subjects have reported lateralized abnormalities in the left hemisphere,[42–46] but no correlation with handedness could be established. It would be advisable for future researches, to include the evaluation of handedness preference and its possible connection with the presence of lateralized abnormalities in violent individual groups.

Comparisons between our previously reported data[10] and the current one, with regard to different visual assigned EEG categories, found a significant difference for the slow EEG. In the current ASPD group, slow EEG activity was decreased and paroxysmal activity increased. However, the results of both researches could potentially be interpreted as evidence of a central nervous system (CNS) dysfunction in the ASPD diagnosis.

QEEG abnormalities observed in these two ASPD groups were different. In this research was found that the ASPD group has an increase of slow activities in the left parietal and both temporal regions, a decrease of alpha energy at the right fronto-temporal, left temporal and parietal derivations, as well as an excess of beta energy in the right frontal and bilateral temporal region of the head. The current study did not replicate our previous findings. QEEG differences between groups could be related to the sample studied and may reveal the implicit heterogeneity present in the ASPD diagnosis. These findings coincide with the hypothesis established, where QEEG abnormalities may be representative of selected samples and could not be replicated in others ASPD groups. The DSM-IV criteria for ASPD are exclusively based on behaviour, not on etiology. It means that the origin of antisocial behaviour could be related to diverse etiologies, and these etiologies are associated with different QEEG profiles as well. An important finding was that the sources of theta slow activity were higher on the left temporal and parietal lobes in ASPD offenders, using LORETA. It agrees with previous studies which found dysfunction of temporal and parietal areas in violent offenders.[47–49] In general, LORETA helped to define the structures related to the increase of slow activity as a sign of a dysfunctional neuronal state, probably secondary to central nervous system injury.

Results of the current research could be considered for developing of statistical classification algorithms in order to discriminate the electrical brain activity between ASPD offenders and individuals without this disorder. This type of methodology has been used in studies for patients' differentiation among those that suffer from Alzheimer's diseases, schizophrenia and attention deficit hyperactivity disorder (ADHD) in adult patients.[50–52] Applications of these modern methods could be useful as an additional tool to improve the objectivity of the ASPD diagnosis.

It is recommended for future research, to involve more homogeneous groups, to use more precise diagnostic criteria, like the PCL-R scale[53] and questionnaire for aggressive behaviour classification, it allows the correct interpretation of electrophysiological findings found in violent subjects.

Conflict of interest

The authors declare no conflict of interest.

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Ethical approval

No ethical approval is needed as it is a short report.

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